WEST Search History

DATE: Friday, May 16, 2003

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L4	chimeric phage and host range	6	L4
DB=PGPB; PLUR=YES; OP=ADJ			
L3	chimeric phage and host range	1	L3
L2	houtzager.in.	1	L2
L1	09882621.an.	0	L1

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 DIS HIST

     (FILE 'HOME' ENTERED AT 16:16:00 ON 16 MAY 2003)

     FILE 'MEDLINE' ENTERED AT 16:16:08 ON 16 MAY 2003
L1            16 S PHAGE AND COAT AND HOST AND MUTANT

=>
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L1 ANSWER 1 OF 16 MEDLINE
AN 2002051177 MEDLINE
DN 21635300 PubMed ID: 11771966
TI Transmembrane domain mediated self-assembly of major **coat** protein subunits from Ff bacteriophage.
AU Melnyk Roman A; Partridge Anthony W; Deber Charles M
CS Division of Structural Biology and Biochemistry. Research Institute, University of Toronto, Hospital for Sick Children, Toronto, Ontario M5G 1X8, Canada.
SO JOURNAL OF MOLECULAR BIOLOGY, (2002 Jan 4) 315 (1) 63-72.
Journal code: 2985088R. ISSN: 0022-2836.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200201
ED Entered STN: 20020125
Last Updated on STN: 20020131
Entered Medline: 20020130
AB The 50-residue major **coat** protein (MCP) of Ff bacteriophage exists as a single-spanning membrane protein in the Escherichia coli **host** inner membrane prior to assembly into lipid-free virions. Here, the molecular bases for the specificity and stoichiometry that govern the protein-protein interactions of MCP in the **host** membrane are investigated in detergent micelles. To address these structural issues, as well as to circumvent viability requirements in **mutants** of the intact protein, peptides corresponding to the effective alpha-helical TM segment of wild-type and **mutant** bacteriophage MCPs were synthesized. Fluorescence resonance energy transfer (FRET) experiments on the dansyl and dabcyl-labeled MCP TM domain peptides in detergent micelles demonstrated that the peptides specifically associate into non-covalent homodimers, as postulated for the biologically relevant membrane-embedded MCP oligomer. MCP peptides labeled with short-range pyrene fluorophores at the N terminus displayed excimer fluorescence consistent with homodimerization occurring in a parallel fashion. Variant peptides synthesized with single substitutions at helix-interactive positions displayed a wide range of dimer/monomer ratios on SDS-PAGE gels, which are interpreted in terms of steric volume, presence or absence of beta-branching, and the effect of polar substituents. The overall results indicate discrete roles for helix-helix interfacial residues as packing recognition elements in the membrane-inserted state, and suggest a possible correlation between **phage** viability and efficacy of MCP TM-TM interactions.

L1 ANSWER 2 OF 16 MEDLINE
AN 2000208552 MEDLINE
DN 20208552 PubMed ID: 10746763
TI A bacteriophage-like particle from Bartonella bacilliformis.
AU Barbian K D; Minnick M F
CS Division of Biological Sciences, The University of Montana, Missoula 59812-4824, USA.
NC AI45534 (NIAID)
SO MICROBIOLOGY, (2000 Mar) 146 (Pt 3) 599-609.
Journal code: 9430468. ISSN: 1350-0872.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200006
ED Entered STN: 20000629
Last Updated on STN: 20000629
Entered Medline: 20000621

AB Bartonella bacilliformis and Bartonella henselae, the respective agents of Oroya fever and cat-scratch disease in humans, are known to produce bacteriophage-like particles (BLPs) that package 14 kbp segments of the **host** chromosome. Data from this study suggest that other Bartonella species including Bartonella quintana, Bartonella doshiae and Bartonella grahamii also contain similar BLPs, as evidenced by the presence of a 14 kbp extrachromosomal DNA element in their genomes, whereas Bartonella elizabethae and Bartonella clarridgeiae do not. A purification scheme utilizing chloroform, DNase I and centrifugation was devised to isolate BLPs from B. bacilliformis. Intact BLPs were observed by transmission electron microscopy and were round to icosahedral in shape and approximately 80 nm in diameter. RFLP and Southern blot analysis of BLP DNA from B. bacilliformis suggest that packaging, while non-selective, is less than the near-random packaging previously reported for the B. henselae **phage**. Data also suggest that the linear, double-stranded BLP DNA molecules have blunt ends with noncovalently closed termini. Packaging of the BLP DNA molecules into a protein **coat** appears to be closely related to nucleic acid synthesis, as unpackaged **phage** DNA is not detectable within the **host** cell. SDS-PAGE analysis of purified BLPs from B. bacilliformis showed three major proteins with apparent molecular masses of 32, 34 and 36 kDa; values that closely correspond to proteins found in B. henselae BLPs. Western blot analysis performed with patient convalescent serum showed that BLP proteins are slightly immunogenic in humans. To determine if BLPs contribute to horizontal gene transfer, **mutants** of B. bacilliformis were generated by allelic exchange with an internal fragment of the 16S-23S rDNA intergenic spacer region and a suicide vector construct, termed pKB1. BLPs from one of the resultant strains were able to package the mutagenized region containing the kanamycin-resistance cassette; however, numerous approaches and attempts at intraspecies transduction using these BLPs were unsuccessful.

L1 ANSWER 3 OF 16 MEDLINE
AN 1999229406 MEDLINE
DN 99229406 PubMed ID: 10212936
TI Historical overview of research on the tobacco mosaic virus genome: genome organization, infectivity and gene manipulation.
CM Erratum in: Philos Trans R Soc Lond B Biol Sci 1999 Dec 29;354(1352):2083
AU Okada Y
CS Department of Bioscience, Teikyo University, Utsunomiya, Japan.
SO PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY OF LONDON. SERIES B: BIOLOGICAL SCIENCES, (1999 Mar 29) 354 (1383) 569-82. Ref: 160
Journal code: 7503623. ISSN: 0962-8436.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199905
ED Entered STN: 19990601
Last Updated on STN: 20000421
Entered Medline: 19990520
AB Early in the development of molecular biology, TMV RNA was widely used as a mRNA [corrected] that could be purified easily, and it contributed much to research on protein synthesis. Also, in the early stages of elucidation of the genetic code, artificially produced TMV **mutants** were widely used and provided the first proof that the genetic code was non-overlapping. In 1982, Goelet et al. determined the complete TMV RNA base sequence of 6395 nucleotides. The four genes (130K, 180K, 30K and **coat** protein) could then be mapped at precise locations in the TMV genome. Furthermore it had become clear, a little earlier, that genes located internally in the genome were expressed via subgenomic mRNAs. The initiation site for assembly of TMV particles was also determined.

However, although TMV contributed so much at the beginning of the development of molecular biology, its influence was replaced by that of Escherichia coli and its **phages** in the next phase. As recombinant DNA technology developed in the 1980s, RNA virus research became more detached from the frontier of molecular biology. To recover from this setback, a gene-manipulation system was needed for RNA viruses. In 1986, two such systems were developed for TMV, using full-length cDNA clones, by Dawson's group and by Okada's group. Thus, reverse genetics could be used to elucidate the basic functions of all proteins encoded by the TMV genome. Identification of the function of the 30K protein was especially important because it was the first evidence that a plant virus possesses a cell-to-cell movement function. Many other plant viruses have since been found to encode comparable 'movement proteins'. TMV thus became the first plant virus for which structures and functions were known for all its genes. At the birth of molecular plant pathology, TMV became a leader again. TMV has also played pioneering roles in many other fields. TMV was the first virus for which the amino acid sequence of the **coat** protein was determined and first virus for which cotranslational disassembly was demonstrated both in vivo and in vitro. It was the first virus for which activation of a resistance gene in a **host** plant was related to the molecular specificity of a product of a viral gene. Also, in the field of plant biotechnology, TMV vectors are among the most promising. Thus, for the 100 years since Beijerinck's work, TMV research has consistently played a leading role in opening up new areas of study, not only in plant pathology, but also in virology, biochemistry, molecular biology, RNA genetics and biotechnology.

L1 ANSWER 4 OF 16 MEDLINE
AN 1998300326 MEDLINE
DN 98300326 PubMed ID: 9636697
TI The major **coat** protein of filamentous bacteriophage f1 specifically pairs in the bacterial cytoplasmic membrane.
AU Haigh N G; Webster R E
CS Department of Biochemistry, Duke University Medical Center, Durham, NC 27710, USA.
NC GM18305 (NIGMS)
SO JOURNAL OF MOLECULAR BIOLOGY, (1998 May 29) 279 (1) 19-29.
Journal code: 2985088R. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199807
ED Entered STN: 19980716
Last Updated on STN: 19980716
Entered Medline: 19980708
AB Filamentous bacteriophage are long, thin single-stranded DNA viruses that infect male strains of Escherichia coli without killing the **host** . Each **phage** contains approximately 2700 copies of the major **coat** protein, pVIII, which exists as a 5.2 kDa cytoplasmic membrane protein prior to incorporation into **phage**. Studies from a number of groups analyzing the behavior of wild-type and **mutant** pVIII in detergents suggested that pVIII might pair under these conditions. In order to test whether pVIII molecules pair in vivo in the cytoplasmic membrane, four plasmidencoded pVIII variants were constructed in which specific residues in the transmembrane region were mutated to cysteine in an attempt to stabilize any pair via disulfide bridges. Variants A35C and I39C were unable to complement **phage** with an amber mutation in gene VIII. The I39C variant was unable to be packaged into **phage** particles even though it was inserted into the membrane. In the case of A35C, the inability to complement was not due to a packaging defect because the variant protein could be packaged into **phage** in the presence of wild-type pVIII. Western blot analysis of cytoplasmic membrane samples revealed that the A35C variant

formed stable disulfide dimers in vivo. Expression of A35C interfered with wild-type **phage** infection, indicating that the assembly machinery may recognize the disulfide dimers in some non-productive way. The results indicate that pVIII may specifically pair along a particular face in the cytoplasmic membrane prior to assembly; however, these pairs must be able to be separated in order for normal assembly to occur.

L1 ANSWER 5 OF 16 MEDLINE
AN 1998026832 MEDLINE
DN 98026832 PubMed ID: 9358153
TI Functional recognition of fragmented operator sites by R17/MS2 **coat** protein, a translational repressor.
AU Fouts D E; True H L; Celander D W
CS Department of Microbiology and College of Medicine, University of Illinois at Urbana-Champaign, B103 Chemical and Life Sciences Laboratory, 601 South Goodwin Avenue, Urbana, IL 61801, USA.
NC GM47854 (NIGMS)
SO NUCLEIC ACIDS RESEARCH, (1997 Nov 15) 25 (22) 4464-73.
Journal code: 0411011. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199801
ED Entered STN: 19980129
Last Updated on STN: 19980129
Entered Medline: 19980114
AB The R17/MS2 **coat** protein serves as a translational repressor of replicase by binding to a 19 nt RNA hairpin containing the Shine-Dalgarno sequence and the initiation codon of the replicase gene. We have explored the structural features of the RNA operator site that are necessary for efficient translational repression by the R17/MS2 **coat** protein in vivo . The R17/MS2 **coat** protein efficiently directs lysogen formation for P22 R17 , a bacteriophage P22 derivative that carries the R17/MS2 RNA operator site within the P22 **phage** ant mRNA. **Phages** were constructed that contain fragmented operator sites such that the Shine-Dalgarno sequence and the initiation codon of the affected gene are not located within the RNA hairpin. The wild-type **coat** protein directs efficient lysogen formation for P22 **phages** that carry several fragmented RNA operator sites, including one in which the Shine-Dalgarno sequence is positioned 4 nt outside the **coat** protein binding site. Neither the wild-type R17/MS2 **coat** protein nor super-repressor **mutants** induce lysogen formation for a P22 **phage** encoding an RNA hairpin at a distance of 9 nt from the Shine-Dalgarno sequence, implying that a discrete region of biological repression is defined by the **coat** protein-RNA hairpin interaction. The assembly of RNA species into capsid structures is not an efficient means whereby the **coat** protein achieves translational repression of target mRNA transcripts. The R17/MS2 **coat** protein exerts translational regulation that extends considerably beyond the natural biological RNA operator site structure; however, the **coat** protein still mediates repression in these constructs by preventing ribosome access to linear sequence determinants of the translational initiation region by the formation of a stable RNA secondary structure. An efficient translational regulatory mechanism in bacteria appears to reside in the ability of proteins to regulate RNA folding states for **host** cell and **phage** mRNAs.

L1 ANSWER 6 OF 16 MEDLINE
AN 95395860 MEDLINE
DN 95395860 PubMed ID: 7666434
TI Packing of **coat** protein amphipathic and transmembrane helices in filamentous bacteriophage M13: role of small residues in protein oligomerization.

AU Williams K A; Glibowicka M; Li Z; Li H; Khan A R; Chen Y M; Wang J; Marvin D A; Deber C M
CS Division of Biochemistry Research, Research Institute Hospital for Sick Children, Toronto, Ontario, Canada.
SO JOURNAL OF MOLECULAR BIOLOGY, (1995 Sep 8) 252 (1) 6-14.
Journal code: 2985088R. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199510
ED Entered STN: 19951020
Last Updated on STN: 19970203
Entered Medline: 19951011
AB Filamentous bacteriophage M13, an important cloning and **phage** display vector, is encapsulated by ca 2700 copies of its 50-residue major **coat** protein (gene 8). This protein occurs as a membrane protein while stably inserted into its E. coli **host** inner membrane, and as a **coat** protein upon assembly and packing onto **phage** DNA in the lipid-free virion. To examine the specific protein-protein interactions underlying these processes, we used a combination of randomized and saturation mutagenesis of the entire gene 8 to assess the susceptibility of each position to mutation. In the resulting library of ca 100 viable M13 **mutants**, "small" residues (Ala,Gly,Ser), which constitute the non-polar face of the N-terminal amphipathic helical segment, and a face of the hydrophobic (effective transmembrane) helical segment, were found to be highly conserved. These results support a model in which **coat** protein packing is stabilized by the presence within each protein subunit of two "oligomerization segments", i.e. specific helical regions with faces rich in small residues which function to promote the close approach of alpha-helices.

L1 ANSWER 7 OF 16 MEDLINE
AN 95271669 MEDLINE
DN 95271669 PubMed ID: 7752244
TI Modifying filamentous **phage** capsid: limits in the size of the major capsid protein.
AU Iannolo G; Minenkova O; Petruzzelli R; Cesareni G
CS Dipartimento di Biologia, Universita di Roma Tor Vergata, Italia.
SO JOURNAL OF MOLECULAR BIOLOGY, (1995 May 12) 248 (4) 835-44.
Journal code: 2985088R. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199506
ED Entered STN: 19950629
Last Updated on STN: 19970203
Entered Medline: 19950620
AB Ff filamentous **phages** are long thin cylindrical structures that infect bacteria displaying the F pilus and replicate without lysing the **host**. These structures are exploited to display peptides by fusing them to the amino terminus of either the bacterial receptor protein (pIII) or the major **coat** protein (pVIII). We have analysed a vast collection of **phage mutants** containing substitutions and insertions in the amino terminus of pVIII to ask whether any chemical group of this solvent exposed region of the **phage** capsid has any key function in the **phage** life cycle. Any of the five amino-terminal residues can be substituted by most amino acids without affecting **phage** assembly suggesting that this region does not play any essential role in morphogenesis. However, a deletion of three residues delta (Gly3Asp4Asp5) results in a **phage** clone with an decreased ability to produce infective particles. By engineering **phages** designed to display peptides by fusion to the amino

terminus of the major **coat** protein we have found that **phage** viability is affected by peptide length while peptide sequence plays a minor "tuning" role. Most peptides of six residues are tolerated irrespective of their sequence while only 40% of the **phages** carrying an amino-terminal extension of eight residues can form infective particles. This fraction drops to 20% and 1% when we attempt to insert peptides 10 and 16 amino acids long. We have used this information to build **phage** libraries where each **phage** displays approximately 2700 copies of a different octapeptide all over the **phage** surface.

L1 ANSWER 8 OF 16 MEDLINE
AN 94112000 MEDLINE
DN 94112000 PubMed ID: 8284225
TI Direct genetic selection for a specific RNA-protein interaction.
AU MacWilliams M P; Celander D W; Gardner J F
CS Department of Microbiology, University of Illinois at Urbana-Champaign 61801.
NC GM27817 (NIGMS)
SO NUCLEIC ACIDS RESEARCH, (1993 Dec 11) 21 (24) 5754-60.
Journal code: 0411011. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199402
ED Entered STN: 19940228
Last Updated on STN: 19990129
Entered Medline: 19940214
AB The decision between lytic and lysogenic development of temperate DNA bacteriophages is determined largely by transcriptional regulation through DNA-binding proteins. To determine whether a heterologous RNA-binding activity could control the developmental fate of a DNA bacteriophage, a derivative of P22 was constructed in which the chosen developmental pathway is regulated by an RNA-binding molecule interacting with its RNA target site located in a **phage** mRNA. In the example presented, lysogenic development of the **phage** relies upon R17 **coat** protein expression in the susceptible **host** cell and the availability of a suitable **coat** protein binding site encoded by the **phage** genome. Through the analysis of **phage** **mutants** that are able to grow lytically in susceptible cells that express the **coat** protein, additional insights were obtained regarding the specific interaction of the R17 **coat** protein with its RNA binding site. This study also suggests a novel and extremely sensitive strategy for selecting RNA-binding activities in vivo.

L1 ANSWER 9 OF 16 MEDLINE
AN 92184779 MEDLINE
DN 92184779 PubMed ID: 1544912
TI Transmembrane region of wild-type and **mutant** M13 **coat** proteins. Conformational role of beta-branched residues.
AU Deber C M; Li Z; Joensson C; Glibowicka M; Xu G Y
CS Division of Biochemistry Research, Hospital for Sick Children, Toronto, Canada.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1992 Mar 15) 267 (8) 5296-300.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199204
ED Entered STN: 19920424
Last Updated on STN: 19970203
Entered Medline: 19920414

AB Although transmembrane (TM) segments of integral membrane proteins are putatively alpha-helical in conformation, beta-sheet promoters (Val, Ile, Thr) often account for approximately 40% of TM residue composition. We are examining the conformational role(s) of these residues, using as a model system the major **coat** protein of the filamentous bacteriophage M13. This 50-residue protein, which is located at the Escherichia coli **host** membrane during **phage** reproduction, contains a prototypic 19-residue hydrophobic midregion (residues 21-39: YIGYAWAMVVVIVGATIGI). Using "Eckstein" site-directed mutagenesis, we have generated several viable M13 **coat** protein **mutants** with beta-branched amino acid substitutions within their TM region. **Mutant coat** proteins, including Ile32----Val (I32V) and Ala27----Thr (A27T), were obtained in milligram quantities by growing M13 **mutant phages** in liter preparations, confirming that these **coat** proteins are capable of assuming their normal biological function(s) in **phage** reproduction. Circular dichroism spectroscopy performed in the membrane-mimetic medium of deoxycholate micelles indicated comparable alpha-helical contents of **mutants** I32V and A27T to wild-type protein. 13C nuclear magnetic resonance experiments with **mutant** A27T demonstrated that the combination of additional beta-branched content and introduction of an -OH substituent induced chemical shift and temperature-dependent changes and influenced the local protein environment at sites up to 12 residues remote from the mutation site. In contrast, **mutant** I32V (of which a salient feature is a mid-TM pentavaline segment) behaved very similarly to wild-type **coat**. These findings are interpreted in terms of the range of TM secondary structure and stability which can be accommodated by viable M13 **coat** protein **mutants**.

L1 ANSWER 10 OF 16 MEDLINE
AN 92062094 MEDLINE
DN 92062094 PubMed ID: 1953741
TI Viable transmembrane region **mutants** of bacteriophage M13 **coat** protein prepared by site-directed mutagenesis.
AU Li Z; Deber C M
CS Research Institute, Hospital for Sick Children, Toronto, Ontario, Canada.
SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1991 Oct 31) 180 (2) 687-93.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199112
ED Entered STN: 19920124
Last Updated on STN: 19970203
Entered Medline: 19911202
AB Bacteriophage M13 **coat** protein - a 50-residue protein located at the E. coli **host** membrane during **phage** reproduction - is subjected to cytoplasmic, membrane-bound, and DNA-interactive environments during the **phage** life cycle. In research to examine the specific features of primary/secondary structure in the effective transmembrane (TM) region of the protein (residues 21-39: YIGYAWAMVVVIVGATIGI) which modulate its capacity to respond conformationally to the progressive influences of these varying environments, we have prepared over two dozen viable **mutant** **phages** with alterations in their **coat** protein TM regions. **Mutants** were obtained through use of site-directed mutagenesis techniques in combination with three "randomized" oligonucleotides which spanned the TM region. No subcloning was required. Among mutations observed were those in which each of the four TM Val residues was changed to Ala, and several with increased Ser or Thr content, including one double Ser **mutant** (G23S-A25S). Polar

substitutions arising at Gly23 and Tyr24-including G23D, Y24H, Y24D and Y24N-suggested that this local segment resides external to the **host** membrane. Milligram quantities of **mutant** **coat** proteins are obtained by growing M13 **mutant** **phages** in liter preparations, with isotopic (e.g., 13C) labelling at desired sites, for subsequent characterization and conformational analysis in membrane-mimetic media.

L1 ANSWER 11 OF 16 MEDLINE
AN 89045639 MEDLINE
DN 89045639 PubMed ID: 3188394
TI Filamentous **phage** assembly: morphogenetically defective **mutants** that do not kill the **host**.
AU Smith G P
CS Division of Biological Sciences, University of Missouri, Columbia 65211.
NC AI20564 (NIAID)
SO VIROLOGY, (1988 Nov) 167 (1) 156-65.
Journal code: 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198812
ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19881214
AB The filamentous **phage** virion is assembled without killing the **host**, by extrusion of the DNA through the envelope and concomitant acquisition of **coat** proteins from the inner membrane. When assembly is blocked, however, intracellular **phage** DNA and gene products accumulate and the **host** is killed. This "cell killing" is largely absent in **phage** fd-tet, which carries a tetracycline-resistance determinant within the origin of minus-strand synthesis; as a result of the replication defect, **phage** DNA does not accumulate to high levels intracellularly when virion assembly is blocked. This allows morphogenetically defective **mutants** except those ablating gene V to be freely propagated in tetracycline-containing medium and studied in the absence of the confounding factor of cell morbidity. Because cultures can be initiated by transfection in the complete absence of input virions, extremely low levels of **phage** production can be assayed. Using this system, I show that genes III, VI, I, and IV are not required to form the complex between viral DNA and gene-V protein that is the intracellular precursor to mature virions; that genes I and/or IV are absolutely (or nearly absolutely) required for assembly; and that mos, a cis-acting sequence previously shown to enhance **phage** yield in some circumstances, is without such effect in others.

L1 ANSWER 12 OF 16 MEDLINE
AN 83225612 MEDLINE
DN 83225612 PubMed ID: 6407198
TI Nonsense **mutants** of the lipid-containing bacteriophage PR4.
AU Davis T N; Cronan J E Jr
NC GM26156 (NIGMS)
SO VIROLOGY, (1983 Apr 30) 126 (2) 600-13.
Journal code: 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198307
ED Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19830708

AB Thirty-three nonsense **mutants** of **phage** PR4 representing 12 complementation groups were isolated. One or two **mutants** of each group were grown on a suppressor-negative (Su-) **host** and characterized by the following criteria (i) proteins synthesized, (ii) level of **phage** DNA synthesis, and (iii) ability to assemble particles. We determined the protein and phospholipid compositions of the particles assembled in an Su- **host**, the presence of DNA in the particles, and the ability of the particles to adsorb to **host** cells. Finally each complementation group was tested for the ability to lyse an Su- **host**. We have identified one protein required for DNA synthesis, five proteins required for proper assembly of the protein **coat** and lipid membrane of the **phage**, two proteins required for stable insertion of DNA into the virion, a protein required for adsorption, a protein required for attachment of the adsorption protein to the virion, and a **phage** -encoded lytic enzyme.

L1 ANSWER 13 OF 16 MEDLINE
AN 82127586 MEDLINE
DN 82127586 PubMed ID: 7036184
TI Filamentous **phage** assembly: membrane insertion of the major **coat** protein.
AU Model P; Russel M; Boeke J D
SO PROGRESS IN CLINICAL AND BIOLOGICAL RESEARCH, (1981) 64 389-400. Journal code: 7605701. ISSN: 0361-7742.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198204
ED Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19820412
AB The assembly of filamentous bacteriophages has been studied in cells infected by wild-type and **mutant phage**; **host** **mutants** defective in bacteriophage assembly have also been isolated. **Phage** assembly takes place at the membrane, and requires insertion of the viral major **coat** protein. We present data on the physiology of this process and on the effects of amino acid sequence variations near to the **coat** protein amino terminus on membrane insertion, processing, and **phage** assembly.

L1 ANSWER 14 OF 16 MEDLINE
AN 81199525 MEDLINE
DN 81199525 PubMed ID: 7015343
TI A mutation downstream from the signal peptidase cleavage site affects cleavage but not membrane insertion of **phage** **coat** protein.
AU Russel M; Model P
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1981 Mar) 78 (3) 1717-21. Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198107
ED Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19810723
AB Morphogenesis of filamentous **phage** includes synthesis of the **phage** major **coat** protein in precursor form, its insertion into the **host** cell plasma membrane, its cleavage to the mature form of the protein, and its assembly there into virions. The

M13 **mutant** am8H1R6 encodes a **coat** protein in which leucine replaces glutamic acid as residue 2 of the mature protein [Boeke, J. D., Russel, M. & Model, P. (1980) J. Mol. Biol. 144, 103-116]. The **coat** protein precursor produced by this variant is a poor substrate for the Escherichia coli signal peptidase both in vivo and in vitro. This pre-**coat** protein, which is eventually processed and assembled into viable **phage** particles, is associated with the membrane fraction of the infected cell. We conclude that the domain recognized by the signal peptidase extends beyond the signal peptide itself. Furthermore, membrane association and signal peptide cleavage can be separated temporally under conditions that permit membrane insertion, cleavage, and **phage** assembly.

L1 ANSWER 15 OF 16 MEDLINE
AN 77140772 MEDLINE
DN 77140772 PubMed ID: 321420
TI Effect of cessation of phospholipid synthesis on the synthesis of a specific membrane-associated bacteriophage protein in Escherichia coli.
AU Cashman J S; Webster R E
SO JOURNAL OF BACTERIOLOGY, (1977 Mar) 129 (3) 1245-9.
Journal code: 2985120R. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197705
ED Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19770512
AB The major **coat** protein of the bacteriophage f1 is synthesized during infection of Escherichia coli and becomes tightly associated with the **host** membrane. This synthesis was studied in conjunction with the strain BB26-36, a **mutant** defective in phospholipid synthesis, to investigate basic questions concerning membrane protein and phospholipid synthesis. **Coat** protein synthesis is decreased in the absence of net phospholipid synthesis. The **coat** protein produced under these conditions is still found tightly associated with the membrane. Resumption of phospholipid synthesis leads to an increase in the synthesis and accumulation of the **coat** protein. Therefore, a correlation between **coat** protein and phospholipid synthesis seems to exist. However, the packaging of **phage** deoxyribonucleic acid into **phage** particles proceeds in the absence of phospholipid synthesis, and the number of **phage** particles produced appears to depend only on the amount of **coat** protein in the membrane.

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TI Comparative studies on the structural proteins of T3 and T7 **phages**.
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AB A comparison of the **coat** protein patterns of the wild types of the related **phages** T3 and T7 on SDS polyacrylamide gel

electrophoresis was carried out. After infection of the nonpermissive **host** with T7 amber **mutants** in genes 7, 11, 12, 13 or 17 and T3 amber **mutants** in genes 11, 12, 13 and 17 respectively, noninfectious, DNA containing particles were produced. The protein pattern, as well as electron microscopy of defective particles of T3 and T7 led to the conclusion that the proteins specified by genes 11, 12, 13 and 17 are tail proteins whereas the proteins coded by genes 7, 8, 10, 14, 15 and 16 enter the head structure. The "serum blocking protein" (gene 17 product) seems to play a different role in the assembly of T3 and T7 tails, since T3 particles defective in gene 17 did not show any tail structure connected with the head whereas T7 particles defective in gene 17 had a tail which looked different from that of the wildtype. Treatment of wildtype particles with ammonium nitrate or sodium pyrophosphate led to morphologically abberrant forms which had partially or completely lost the hexagonal head structure. After treatment with ammonium nitrate ball-like structures were obtained, both for T3 and T7. However, in the case of T3 these abberrant forms contained the proteins specified by genes 7, 8 and 10 whereas those derived from T7 contained only the proteins specified by genes 8 and 10. Sodium pyrophosphate treatment of T3 and T7 wildtype particles led to a release of the **phage** tails. The isolated tails were examined by electron microscopy thus revealing for the first time a detailed substructure of the T3 and T7 **phage** tails. In order to find out more about the tail proteins, absorption experiments with isolated bacterial cell walls were carried out.